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arterial insufficiency and immunosuppression. The goal of medicinal leech therapy is to relieve venous congestion by allowing venous outflow [1], which is accomplished as a result of contact with the chemical constituents of leech saliva. At the time of the bite, saliva containing hirudin (an anticoagulant), a vasodilator, hyaluronidase, and other substances are secreted onto the wound, resulting in prolonged venous bleeding [1]. In some cases, wounds can bleed up to 48 hours [1, 2, 4]. Wound infections and bleeding requiring transfusions are the most common complications.

The most common wound infections associated with medicinal leech therapy are reportedly due to *Aeromonas hydrophila* [4–6], a gram-negative rod that is part of the normal gut flora of leeches [1, 3, 5] and plays an important role in their digestive capability. We report what we believe is the first case of *Vibrio fluvialis* wound infection associated with medicinal leech therapy.

A 67-year-old man with squamous cell carcinoma of the mouth underwent anterior resection of the floor of the mouth with pedicled myocutaneous flap repair. On the first day after surgery, he developed venous congestion due to pedicle compromise. His clinical status prevented flap revision in the operating room. In an attempt to salvage the flap, leech therapy was started. Two leeches were applied to the cutaneous portion of the flap twice a day. With each application, good capillary refill was observed. This therapy was maintained for 4 days, at which time the flap remained pink in the absence of leech therapy.

On the sixth post-operative day, purulent drainage, fever (temperature, 38.5°C), and leukocytosis were noted. Culture of the wound subsequently yielded *V. fluvialis*. Treatment with iv doxycycline (100 mg b.i.d.) was initiated, and the infection resolved after 10 days. Unfortunately, the skin overlying the flap became necrotic, and subsequently, a split-thickness skin graft and a local advancement rotation flap were required. A culture of the media in which the leeches were stored also yielded *V. fluvialis*. Since the storage media were prepared under sterile conditions by the hospital pharmacy, we concluded that the most likely source of *V. fluvialis* was the gut flora of the leeches.

Acute diarrheal illness caused by *Vibrio cholerae* or *Vibrio parahaemolyticus* is the most common manifestation of infection with *Vibrio* species. The species most often associated with soft-tissue infections are *V. alginolyticus*, *V. damsela*, and *V. vulnificus* [7].

V. fluvialis, formerly called EF-6, is a halophilic organism [7, 8]. It has been associated with acute diarrheal illness worldwide, as well as along the coastal areas of the United States [8–10].

***Helicobacter cinaedi* Bacteremia Associated with Localized Pain but Not with Cellulitis**

Burman et al. [1] and Kiehlbauch and colleagues [2] recently described several cases of *Helicobacter cinaedi* bacteremia and cellulitis. Most patients presented with fever and cutaneous mani-

festations. There have been two previous reports of wound infection associated with *V. fluvialis* [8, 9]. However, these cases were not associated with medicinal leech therapy.

In summary, wound infection is one of the complications of medicinal leech therapy. The findings of our case suggest that, in addition to *A. hydrophila*, *V. fluvialis* should be considered as a possible etiologic agent of such infections. Doxycycline is effective in the treatment of *V. fluvialis* infection and may be considered the drug of choice. Alternative therapeutic agents are second-generation cephalosporins and the fluoroquinolones that have been used to treat infections caused by other *Vibrio* species [7].

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festations. We report a case of *H. cinaedi* bacteremia with fever and involvement of soft tissue of the leg but without cellulitis or other visible skin infection.

A 41-year-old HIV-seropositive homosexual man with low CD4 cell counts ($<0.05 \times 10^9/L$) for >1 year presented with a 1-month history of pain in his right lower leg that was first felt only when walking and later continuously; he had previously been in good general condition and had no history of AIDS-defining conditions. He also had fever and a generalized rash. Physical examination revealed tenderness of the right lower leg without swelling, erythema, or signs of arthritis of the ankle joint. Fever and rash were initially attributed to co-trimoxazole therapy, but because his fever persisted after discontinuation of this therapy, further investigations were carried out.

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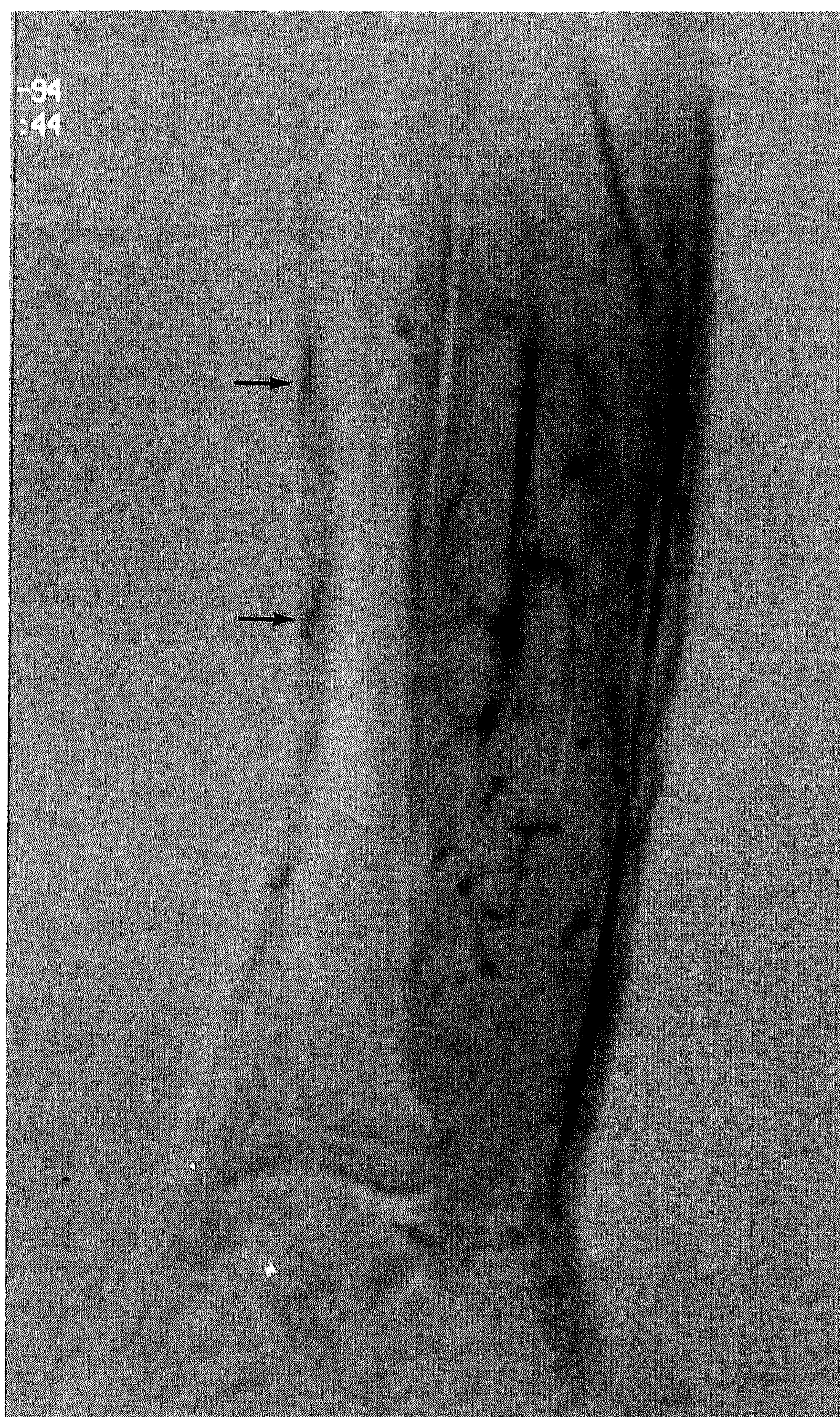


Figure 1. MRI of the right lower leg of a patient with *Helicobacter cinaedi* bacteremia with localized pain but without cellulitis that shows increased soft-tissue vascular structures as well as intramedullary vascular structures in the tibia (arrows).

A radiograph of the right lower leg did not show any abnormality, but three-phase skeletal scintigraphy revealed increased perfusion and pools of blood in the distal right lower leg with normal delayed images. These findings were suggestive of soft-tissue pathology without osseous involvement. Furthermore, scintigraphy with indium-111-labeled human IgG, a procedure for imaging inflammation and infection [3], showed markedly increased activity in the soft tissues of the right lower leg without involvement of the bony structures. Echography of this area failed to demonstrate any circumscribed abnormalities, but increased arteriolar and venous flow was noticed with use of the Doppler mode. MRIs of the right lower leg, with and without contrast medium (gadolinium; Magnevist, Schering, Weesp, the Netherlands) showed increased soft-tissue

vascular structures as well as intramedullary vascular structures in the tibia (figure 1). MRI with the fast spin-echo sequence FLASH (fast low-angle shot; a sensitive method for the detection of vascular structures) also revealed these intramedullary vascular structures, and a high local signal indicated a slow blood flow.

Bacillary angiomatosis was suspected, and the patient was treated empirically with oral erythromycin (500 mg q.i.d.). No material was obtained from the lesion for culture. This treatment did not result in any clinical effect, and *Bartonella henselae* infection could not be demonstrated by serology and PCR analysis of peripheral blood. Routine blood cultures (7-day cycle) were negative (BACTEC 9240, Becton Dickinson Benelux, Erembodegem-Aalst, Belgium). Fever and pain persisted for several weeks; no specific cause for these symptoms could be detected.

Several weeks later, cultures of two blood specimens drawn during an outpatient visit became positive for gram-negative *Campylobacter*-like organisms after 5 days of incubation. Subcultures became positive after 3 days of growth under micro-aerophilic conditions at 37°C. No growth was observed at 25°C and 42°C. The organisms were identified as *H. cinaedi* by numerical analysis of gel electrophoretic protein profiles [4]. The isolate was susceptible in vitro to cefazolin and tetracycline but was resistant to erythromycin, ciprofloxacin, and ceftriaxone. The patient was treated with intravenous cefazolin (1 g q.i.d.), after which his fever disappeared and the pain in the right lower leg subsided. After 2 weeks, treatment was switched to doxycycline (200 mg daily), and he was discharged. He presented again with fever and pain in his leg. The symptoms disappeared after the medication was switched to tetracycline and did not recur after a 4-week course of this therapy (500 mg q.i.d.) was completed. After 6 months, no recurrence of this infection was observed. It was surprising that the doxycycline therapy was a clinical failure.

Our case confirms previous findings [1, 2] that erythromycin and ciprofloxacin are not the drugs of choice for treatment of infection with this particular strain. Moreover, our isolate was resistant to ceftriaxone but was susceptible in vitro to cefazolin. Cefazolin also proved to be clinically useful.

In their series Burman et al. [1] reported that prolonged bacteremia and skin lesions were seen in cases of *H. cinaedi* bacteremia, thus suggesting that endovascular infection may be a feature of *H. cinaedi* bacteremia (as has been suggested for *Campylobacter fetus* bacteremia [5, 6]). The increased vascular structures seen on the MRIs of our patient support this suggestion.

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